wide daily variations, since the concentration within the red cell is constant for the cell's 120-day life span. Although the Center for Disease Control guidelines considered an EP value of less than 60 μg per 100 ml of whole blood as normal, studies indicate that the upper limit of normal in children is closer to 45 μg per 100 ml of whole blood.[5] So the group of children with blood lead levels between 30 and 50 μg per 100 ml and EP values less than 60 μg per 100 ml should not be ignored.

In general, projects such as the one in California, will find that most of the children with elevated lead levels and EP values are asymptomatic and the elevations are not pronounced. How to relate the blood level or EP value to the risk of clinical toxicity is the dilemma of assigning a threshold of toxicity. Certainly it is the neuron, rather than the red cell precursor with deranged heme synthesis, that is the chief concern. The manifestation of neurologic damage in asymptomatic children will not be apparent for some years after the period of increased ingestion and is likely to consist of learning or behavioral disturbances. In the most relevant, though not a perfect study, de la Burde and co-workers found that poor academic performance, primarily due to behavioral problems, was more frequent among children with increased exposure to lead during early childhood.[6]

The lead problem in children is a frustrating one. The full impact is not known. Safe elevations cannot be confidently given. Successful management depends on socioeconomic and cultural factors as well as medical practice. Each community must consider whether there is a population of children at risk. Then a screening program must find them. The legacy of old, substandard housing and the likelihood of continued pollution will make lead a problem for a long time.

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REFERENCES

1. Childhood Lead Based Paint Poisoning Prevention Program—Reported Data—FY 1975. Atlanta, Center for Disease Control, US Dept of HEW, Public Health Service, Jan 1976
2. Needleman HL, Davidson I, Sewell EM, et al: Subclinical lead exposure in Philadelphia school children—Identification by dentine lead analysis. N Engl J Med 290:245-248, Jan 1974
3. Sayre JW, Charney E, Vostal J, et al: House and hand dust as a potential source of childhood lead exposure. Am J Dis Child 127:167-170, Feb 1974
4. Rabinowitz MB, Wetherill GW, Kopple JD: Kinetic analysis of lead metabolism in healthy humans. J Clin Invest 58:260-270, Aug 1976
5. Piomelli S, Davidow B, Guinee VF: Letter to the editor. Pediatrics 52:304, Aug 1973
6. de la Burde B, Choate MS: Early asymptomatic lead exposure and development at school age. J Pediatr 87:638-642, Oct 1975

PSRO — Update 1977

THIS IS THE FOURTH in a series of more-or-less annual editorial reports on Professional Standards Review Organization (PSRO) developments. One senses that during the past year slow but steady progress has been made toward implementing the complex and potentially very important PSRO law. It is expected that by the end of this federal fiscal year (30 September 1977) there will be 119 conditional PSRO's in place and 70 planning PSRO's proceeding toward conditional status, with only a few of the designated areas remaining unfunded. In a very few areas no PSRO has yet developed. The present law has extended the time professional organizations will have priority in establishing PSRO's until 1 January 1978. The hope has been expressed by the government that it will not be necessary to invoke the alternative means provided by the law to create PSRO's in those areas which have not been able to establish one.

Much more remains to be resolved at the federal level. The tension between the law's dual priorities of cost control and quality assurance continues, with neither having gained a clear ascendency within the federal bureaucracy. This is probably as it ought to be. Too much emphasis on one adversely affects the other. Attempts are underway at the bureaucratic and legislative levels to clarify the relationships of PSRO's to the state review mechanisms for Medicaid programs and to the federally mandated medical review boards of the national End Stage Renal Disease (ESRD) program. The confidentiality issue also has not been settled. The Privacy Protection Study Commission, created by Congress in 1974, has called upon the Department of Health, Education, and Welfare to require hospitals to adopt procedures to guard the privacy of medical records as a condition of qualifying for Medicare and Medicaid reimbursement. But what actually will be done to protect the privacy of physicians and their patients remains to be seen. Meanwhile there is talk of developing patient, physician and institutional profiles for analysis to make comparisons

of patterns of care by similar practitioners and institutions for similar patients, and so to identify exceptional patterns which will allow PSRO's and hospitals to use their resources in the most efficient ways.

All of this appears to be by way of preparation for National Health Insurance which is envisioned as requiring effective mechanisms of quality and cost control. It is noteworthy, and significant, that of all the control mechanisms being tried by government, the PSRO mechanism emerges as the only one clearly accountable to the medical profession. In times such as these, this should be a powerful incentive to physicians to make PSRO's work and work well in the best interests of all concerned.

—MSMW

Rheumatoid Synovium

Its Proliferation, Heterogeneity and Polarization

All of the changes observed suggest that a viral agent may form the basis for these alterations [in rheumatoid synovium]. However, studies to date have failed to isolate or identify the putative causative virus and the search continues.

THESE SENTENCES end the opening abstract in the lucid review of the biology of the rheumatoid synovial membrane by Haselwood and Castles published in this issue. It is a good review and an accurate summary. The truth may be that a virus is involved initially, but that its role in producing the proliferative and destructive lesion of rheumatoid arthritis may be minimal. It may even be true that virus infection may be (as is one of the possibilities in multiple-choice examinations) "true, true and not related." If a virus is found not to be clearly *the* cause of rheumatoid arthritis, it may not be the inadequacy of our laboratory techniques so much as it is the erroneous assumption forming the basis of our approach to most diseases in this day and time—that is, the assumption that if we search hard enough, a single metabolic (often genetic) defect or underlying infectious agent capable of replication and derived from our environment eventually will be identified as the initiating factor of each ailment listed in our textbooks of medicine.

Common to all rheumatoid synovial tissues is a striking degree of nonmalignant proliferation, cellular heterogeneity and polarization. Every physician should take the opportunity to go into the operating room and observe an orthopaedic colleague carry out a synovectomy and compare the mass of brown-red tissue removed with the paper-thin transparent membrane which forms the "synovial membrane" of normal joints. Weight of the pathologic tissue may exceed that of the normal by 500-fold and have a similarly increased synthetic and degradative capacity as well.

The morphologic heterogeneity of the rheumatoid synovial lesion is most clearly evident by looking at thin sections by high resolution transmission phase microscopy.[1] Almost every cell has a morphology different from its neighbor. Multinuclear cells, lymphocytes, the other plasma cells and endothelial cells are most easily recognized. The majority of cells are distinguished by variegate shape, much cytoplasm and location amidst a low-density matrix of collagen fibrils and ground substance. Observation of the pannus/cartilage junction[2-4] shows that some of these cells have multiple thin cytoplasmic extensions penetrating cartilage matrix, and some have dense accumulations of rough endoplasmic reticulum. But because of the enormous numbers of these cells, who is to say that the apparent synovial "A" cells are not, instead, marrow-derived macrophages, or that both types of cell are not, in fact, synovial cells altered beyond functional recognition by lymphokines, connective tissue activating peptides or as yet unidentified humoral substances associated with the rheumatoid process? Dayer and co-workers[5] and Werb and associates,[6] using methods for study of function of dissociated adherent rheumatoid cells in culture, have focused interest on a dendritic or stellate cell with long, spiny cell processes extending radially from the nuclear area. This cell, which has morphologic characteristics similar to adherent cells of mouse spleen studied by Steinman and Cohn,[7] is present along with cells appearing as fibroblasts and with round cells (? macrophages) in these cultures. Interestingly, the number of dendritic cells in proportion to other cells in culture is often increased in cultures that synthesize and release large amounts of collagenase and prostaglandins. The argument for studying dissociated, adherent